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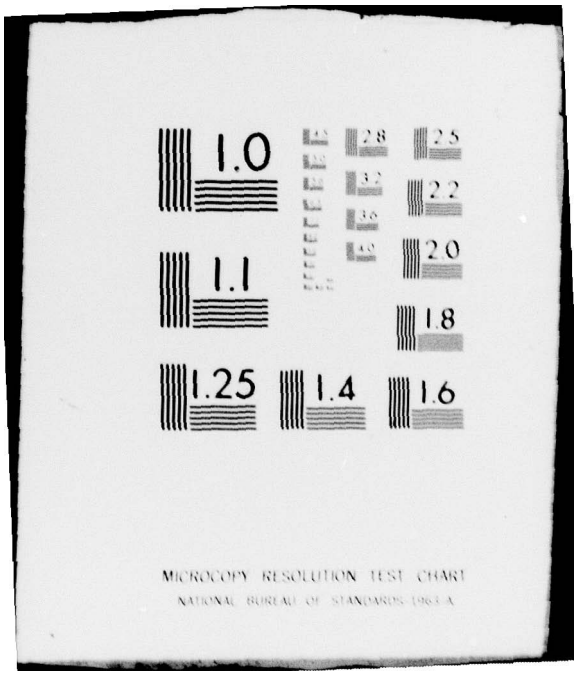
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The annual Symposium of the Fleischner Society is the major event of the year in chest radiology. The society's multidisciplinary approach is conducive to important papers and symposia concerning chest disease that may bear only indirectly on the practice of radiology. While there was no single unifying theme, the meeting was highlighted by Sackner's review of his work on the mucociliary transport mechanism in asthma, Weibel's impressive lecture on the microstructure of the lung and its relationship to pulmonary physiology, and West's plans for future experiments during weightlessness.

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THE FLEISCHNER SOCIETY SYMPOSIUM

"Dedicated to advancing knowledge of the normal and diseased chest" is the creed of the Fleischner Society, which held its 9th annual symposium in Stockholm, Sweden, 11-13 June 1979.

The Society was formed ten years ago following the death of Felix Fleischner, an outstanding radiologist, whose primary interest was in pulmonary problems. Although the Fleischner Society consists mainly of radiologists, a multidisciplinary approach to diseases of the chest is stressed, and many of the speakers were from medical disciplines other than radiology. This broadly based approach to thoracic disorders gave the symposium medical depth that is often lacking in meetings limited to a single medical discipline.

The first two papers covered different aspects of radiologic anatomy; each was characterized by a careful analysis of routine chest radiographs. The first, by John Peter Lavender (Hammersmith Hospital, London), described the normal vascular anatomy of the pulmonary hila by comparing plain radiographs, tomograms, pulmonary angiograms, and anatomic specimens. He also discussed changes that occurred in hilar arteries and veins under conditions of raised left atrial pressure, pulmonary hypertension and chronic lung disease.

The second paper, by John H.M. Austin (Columbia Presbyterian Medical Center, New York), described the effects of slight obliquity on the lateral chest radiograph. Minor degrees of obliquity are commonplace, as it is not expected that a true lateral projection can be made of each patient on every examination. Austin's first point was that observations and measurements on the lateral projection of the chest varied considerably with slight degrees of obliquity. These measurements are, therefore, not valid under those conditions. There are, however, definite advantages to slightly oblique lateral projections as some structures are seen to better advantage than they are on a true lateral. Those structures best seen with a slight right anterior obliquity are the epicardial fat stripe, the right and left pulmonary arteries, the origins of the upper lobe bronchi, and the posterior wall of the bronchus intermedius. On the other hand the azygous vein and posterior tracheal wall tend to be better visualized in a slight right posterior obliquity than in the true lateral projection. Austin was questioned from the audience as to whether he would advocate a routine slight obliquity instead of a true lateral projection, to which he replied in the negative. However, he recommended that in certain cases a repeat examination in the lateral projection with a slight obliquity would be better for visualization of the anatomy and pathology.

R. Fraser (Univ. of Alabama, Birmingham, Alabama) analyzed the Roentgenologic patterns of atelectasis. Initially Fraser noted that the terminology is somewhat controversial and suggested that the term atelectasis be used in its broadest sense, that is to denote diminished air within the lung associated with reduced volume. He then described four types of atelectasis; resorption, passive, cicatrization, and adhesive. The first is the most common and is caused by the resorption of gas from the alveoli in bronchial obstruction. Fraser noted, however, that in resorption atelectasis on a segmental rather than a lobar basis, air drift between the segments will keep the lung expanded despite the obstruction. Therefore, if segmental atelectasis is present (seen most commonly in infection), collateral air drift must not have occurred. Passive or relaxation atelectasis occurs because air or fluid occupies the pleural space thereby reducing the expansile properties of the chest wall. The lung has a natural tendency to collapse and will do so if removed from the chest. Under normal conditions the tendency for the lung to collapse is opposed by the chest wall, and these two forces, the tendencies of the lung to collapse and the chest wall to expand, are equal and opposite. Cicatrization is caused by reduced compliance and occurs with various types of pulmonary fibrosis. The fourth type of atelectasis described by Fraser, which he calls adhesive atelectasis, depends upon forces acting at the air-tissue interface of the alveolar wall. Any disorder that causes reduction in surfactant diminishes the surface tension at the air-tissue interface and atelectasis can occur. From the clinical point of view Fraser reemphasized that even if there is complete obstruction of a segmental bronchus, radiographs made in inspiration and particularly in expiration may demonstrate air trapping instead of collapse because of collateral air drift.

A paper delivered by Marvin A. Sackner (Univ. of Miami, Florida), describing mucociliary clearance in asthma, was a good example of the multidisciplinary approach to the problems of chest disease common to Fleischner symposia. The work described is not directly related to chest radiology but is basic to pulmonary physiology and the understanding of asthma. The mucociliary apparatus of the airways removes inhaled particles from the mucosa of the trachea and bronchi. There is an optimum interaction between cilia and mucous with the result that surface transport velocity appears to increase from the peripheral towards the central airways. Asthmatics typically demonstrate mucous gland hypertrophy and increased secretions, in addition to denuded ciliated epithelium.

Sackner used radio-opaque Teflon discs in intact dogs and followed their movement by fluoroscopy and video tape. An asthmatic reaction was induced in the dogs by an extract of ascaris. In the animals that responded to the antigenic challenge with bronchospasm,

tracheal mucous velocity decreased and was still significantly decreased after the bronchospasm was no longer detectable.

The possible involvement of a chemical mediator of anaphylaxis was evaluated by bronchial provocation with histamine and acetylcholine solutions that induced bronchospasm comparable to that observed in the antigen challenged dogs. There was, however, no effect on tracheal mucous velocity. Therefore, Sackner concluded that 1) tracheal mucous transport is impaired after inhalation of a specific antigen in allergic dogs, 2) mucociliary dysfunction is unrelated to the presence of bronchospasm, and 3) abnormal mucous transport may be related to the release of slow reactive substance (SRS-A). Sackner then went on to describe recent experiments in six young asthmatic patients during spontaneous acute asthmatic attacks. As in the canine experiments, the human studies suggested that in antigen-induced bronchospasm, patients with allergic bronchial asthma demonstrated a rapid decrease in mucous transport independent of bronchospasm and presumably related to the release of chemical medicators during the asthmatic response.

William M. Thurlbeck (Winnipeg General Hospital, Winnipeg, Canada) discussed the definitions, morphology, pathophysiology, and radiology of chronic airway obstruction. Bronchial air flow in expiration is directly related to the pressure and inversely related to the resistance. Air flow can be reduced by diminishing pressure that occurs in diseases such as emphysema with its loss of pulmonary recoil. Air flow can also be reduced by increasing airway obstruction, and there are many diseases that cause partially obstructing lesions within the airway. Thurlbeck then described and analyzed the clinical and pathological aspects of four kinds of emphysema; panacinar, centrilobular, paraseptal, and irregular.

In describing airways obstruction in small airways, that is those less than 2 mm in diameter, Thurlbeck suggested that inflammation is the most important cause of minimal chronic airways obstruction. Since the resistance in the small airways is low and the total cross section of these airways is very large, it follows that widespread narrowing of the small airways would be required before a significant change would be noted in the total resistance or in the alteration of expiratory flow rates. Therefore the usual pulmonary function tests are of little value. An experimental model to study small airways would be of great help but as yet is unavailable.

In discussing chronic bronchitis Thurlbeck initially made clear that this disease by definition is a clinical not a pathological or radiological entity. Chronic bronchitis is defined as the

chronic excessive secretion of intrabronchial mucous. From a morphological point of view the essential change is hypertrophy of bronchial mucous glands. This enlargement is usually expressed as the Reid index, that is, the ratio of bronchial mucous gland size to wall thickness. This ratio correlates roughly with clinical chronic bronchitis. Grossly one finds excessive intrabronchial mucous; microscopically, in addition to bronchial gland hypertrophy, there is an increased proportion of smooth muscle, edema, congestion, and chronic inflammation of the bronchial walls. Thurlbeck emphasized that it is important to separate chronic bronchitis (mucous hypersecretion) from organic airway narrowing. The latter occurs when emphysema complicates bronchitis.

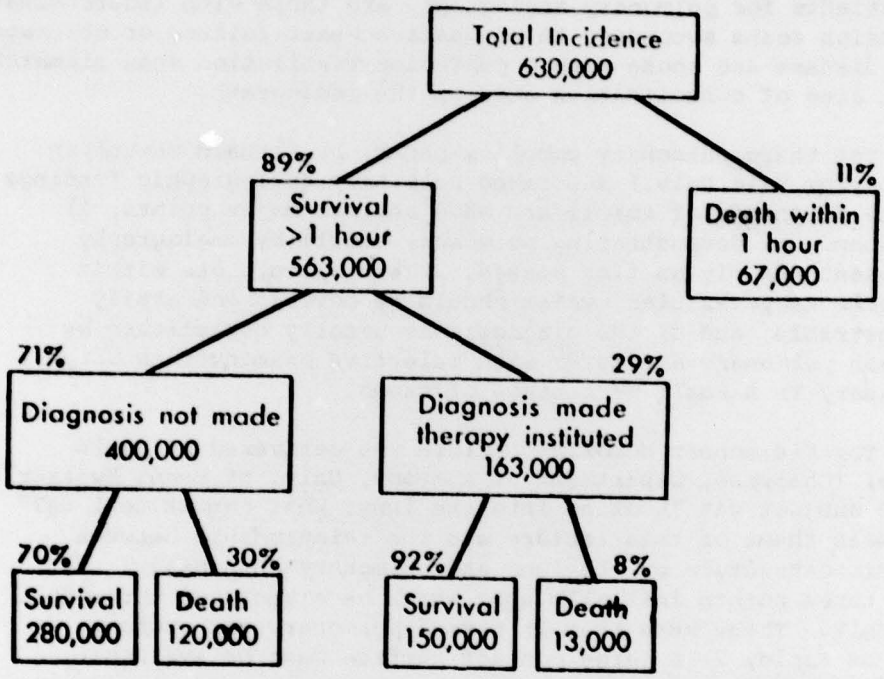
In the last part of his discussion, Thurlbeck correlated radiographic criteria with the diagnosis of emphysema. The only criteria of significant value is a decrease in pulmonary vascularity. He emphasized that only moderately far advanced emphysema can be detected radiographically.

Another subject discussed in depth, by three authors in separate papers, was the diagnosis of pulmonary embolism by clinical, radiographic, radionuclide, and angiographic criteria. Pulmonary embolism is the third most common cause of death in the US. Some of the victims die very quickly and are therefore considered unsalvagable by medical means. For the others, quick and accurate diagnosis is of considerable importance, inasmuch as mortality in the untreated group is much greater than in the treated group—see Chart I.

The first paper by John F. Murray (Univ. of California, San Francisco) emphasized clinical criteria for the diagnosis of pulmonary embolism. The problem is a massive one as approximately 630,000 persons are affected each year in the US alone. The situation is complicated because anticoagulation therapy for pulmonary embolism is associated with a significant morbidity and mortality in itself. Furthermore, patients immobilized for other causes have a significant percentage of thromboembolic disease in the lower leg, and of these approximately 10% will develop pulmonary emboli. For example, of those patients immobilized for myocardial infarction, approximately 25% develop thromboembolic diseases of the lower leg, while of those immobilized for some types of orthopedic surgery approximately 75% will develop thrombi in the lower leg. There are a number of diagnostic procedures used to establish the presence of pulmonary emboli, but only one, pulmonary angiography, is reasonably specific.

The second paper, by Alexander Gottschalk (Yale Univ., New Haven, Conn.), described radionuclide perfusion and ventilation

CHART I



Estimated incidence and outcome of pulmonary embolism in the United States.

scans used for the diagnosis of pulmonary emboli. While a normal perfusion scan essentially rules out pulmonary emboli, a positive perfusion scan may be the result of several different causes. The major diagnostic problem is the selection of patients for pulmonary angiography (see Chart II). The most common groups of patients for pulmonary angiography are those with indeterminate perfusion scans secondary to congestive heart failure or obstructive lung disease and those with a perfusion-ventilation scan mismatch in an area of consolidation seen on the radiograph.

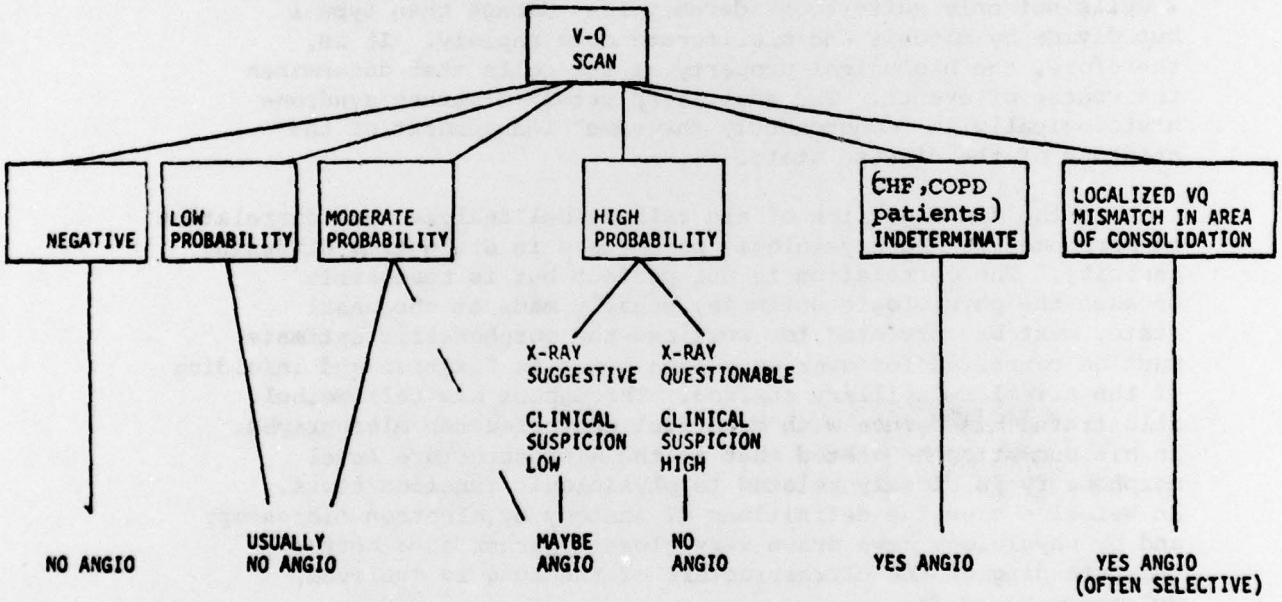
The third pulmonary embolism paper, by Richard Greenspan (also from Yale Univ.) described pulmonary angiographic findings in the diagnosis of emboli and made several major points; 1) the chance of demonstrating pulmonary emboli by angiography decreases sharply as time passes, 2) the blood clots within the pulmonary vascular system should be obvious and easily demonstrable, and 3) the diagnosis is usually established by a flush pulmonary angiogram with selective examinations only necessary in a small percentage of cases.

The Fleischner Memorial Lecture was delivered by Ewald Weibel (Chairman, Department of Anatomy, Univ. of Bern, Switzerland), whose subject was "Looking into the lung: What can it tell us?" The main theme of this lecture was the relationship between the microstructure of the lung and pulmonary physiology. He made three points initially upon which he elaborated throughout his talk. These were that 1) normal pulmonary gas exchange must be rapid, 2) a large contact surface must be available, and 3) the supporting tissue for the lung must represent a very thin barrier for normal gas exchange to take place. He then described the supporting structures, which are peripheral to the lung and connect the lung to the pleura or are axial through the pulmonary lobules with connecting fibers between the two systems.

Electron microscopy of the alveolar-capillary membrane reveals supporting interstitial tissue on one side only while the other side is the very thin two-cell layer alveolar-capillary interface for gas exchange. This alveolar-capillary interface is continuously bathed in a fluid containing a phospholipid, surfactant, that lowers the surface tension almost to zero. Surface tension must remain very low for the thin-walled alveolar spaces to remain open. A reduction in surfactant leads to atelectasis. Therefore, small amounts of fluid can collect in the interstition without affecting gas exchange as long as the surfactant mechanism is intact. On the thin side the two-cell layer consists of the capillary endothelium and the epithelium on the alveolar side. The cells are either type 1 with cytoplasmic extensions

CHART II

Indications for Angiography in patients suspected of having pulmonary emboli



through which gas exchange takes place or type 2 cells which are secretory and responsible for elaborating the surfactant.

Weibel then turned to a common clinical situation, that of the adult respiratory-distress syndrome. He described an exudative phase of one to four days during which the cells are damaged and a proliferative phase after the first four days during which cell repair takes place. The ability of the alveolar-capillary membrane to exchange gas is lost rapidly under conditions of the adult respiratory-distress syndrome and only regained slowly. Type 1 cells are destroyed to a much greater extent than type 2 cells. This leads to the deposition of blood products in the alveolar spaces and along the alveolar-capillary membrane. Fibrin deposition leads to the well-known hyaline membranes. In the proliferate phase gas exchange is inhibited by the replacement of the type 1 cells by large, cuboidal type 2 cells. The type 2 cells not only suffer considerably less damage than type 1 but divide by mitosis and proliferate more rapidly. It is, therefore, the biological property of the cells that determines the course of events. The adult respiratory-distress syndrome histologically is "monotonously the same" independent of the etiology of the disease state.

In the final portion of his talk Weibel analyzed the correlation of morphometric and physiologic parameters in evaluating diffusing capacity. The correlation is not perfect but is reasonable because the physiologic estimate, usually made at the basal state, must be corrected for work and the morphometric estimate must be corrected for over expansion owing to fixation and infolding of the alveolar-capillary surface. Throughout his talk Weibel illustrated his points with beautiful photoelectron micrographs. In his summation he stated that at the microstructure level morphometry is closely related to physiologic function tests. In Weibel's view the definitions of anatomy by electron microscopy and by physiology have drawn very close together as a better understanding of the ultrastructure of the lung is achieved.

Pulmonary blood flow was discussed by two authors, Morris Simon (Harvard Univ. and Beth Israel Hospital, Boston) and Eric Milne (Univ. of California, Irvine). Both of these investigators have spent many years relating the physical laws of flow and pressure to pulmonary physiology and the chest radiograph. It is well known that in the erect human, pulmonary arterial and venous pressure is greater at the lung base than at the lung apex, but Simon reminded the audience that the difference is much larger than one expects. If the diameter of the upper lobe vessels is only one third that of the lower, the ratio of cross-sectional areas is one to nine and of lumenal areas is one to thirty, so that normally flow through the lower lobes is considerably

greater than through the upper lobes. Simon went on to state that blood flow in pulmonary vessels tend to follow Poiseuille's law. For clinical radiology this can be simplified into the statement that flow is proportional to the pressure and to the fourth power of the diameter of the vessel. Velocity will remain constant in the pulmonary vascular system, but the size of the vessels adjust to compensate for differences in flow. This is independent of pulmonary arterial pressure. Clearly in the larger vessels the velocity will be much greater than in the capillary bed. In the capillary bed, because of its vast area, the flow is much slower in spite of the markedly decreased resistance.

Milne's discussion of cardio-pulmonary hemodynamics was similar. His major point was that pulmonary blood flow and pulmonary vascular bed capacity do not have a fixed relationship. The potential capacity of the pulmonary vascular bed is considerably greater than the flow during ordinary life and work. For the entire capacity of the pulmonary vascular bed to be utilized, the organism must be exercised to the point of exhaustion. This concept is to Milne a unifying one that allows him to explain changes in blood flow distribution as observed on the chest radiograph.

Milne made another major point related to pulmonary alveolar pressure. In a number of diseases it is necessary to ventilate the lungs with positive pressure in order to provide sufficient oxygenation. Increased alveolar pressure due to positive pressure ventilation may actually diminish pulmonary capillary blood-flow. The increased alveolar pressure, artificially induced, may indeed increase capillary pressure sufficiently to cause right heart failure.

The correlation between structure and function in disease from chest radiographs and radioisotope scans was discussed by J.M.B. Hughes, J.P. Lavender and R.E. Steiner (Hammersmith Hospital, London). Hughes discussed the relative advantages and disadvantages of ^{81m}Kr (See *ESN* 33-4:159) and ^{133}Xe for radioisotope lung ventilation scans. Both are insoluable gases, but the gamma energy emission of 190 keV of ^{81m}Kr is much more suitable for the gamma camera than the lower energy of ^{133}Xe (80 keV). The dose delivered with ^{81m}Kr is approximately 10% of the dose delivered when using ^{133}Xe, largely because of the short (13 sec) half life of the krypton. The relatively high energy of ^{81m}Kr, in addition, does not interfere with the ^{99m}Tc perfusion scans. The major disadvantage of ^{81m}Kr is that its immediate parent ^{81}Rb with a half life of 4.5 hours, is only available from a cyclotron. The nearby cyclotron makes ^{81}Rb

available to the Hammersmith Hospital approximately three days a week, but the isotope can be and has been shipped all over Europe for medical purposes. Hughes then went on to explain in detail another major advantage of ^{81m}Kr, related to its very short half life in comparison to ^{133}Xe. The quantity of ^{133}Xe breathed is proportional to lung volume but this is not the case with ^{81m}Kr. If ^{133}Xe is inhaled with each breath and a steady state achieved, the "arrival" and "removal" must be analyzed. The arrival is a function of both ventilation and the concentration of the gas, while the removal is a function of the ventilation washout, the concentration and the isotopic decay. Because of the very short half life of the ^{81m}Kr, the ventilation washout and concentration portion of the equation can be eliminated as the krypton decays before it fully mixes with the residual gas in the lung. Therefore, each breath of ^{81m}Kr reflects its arrival in the lung, that is, the distribution of inspired ventilation.

Lavender and Steiner then shared the podium in describing a series of cases. Steiner analyzed the chest radiographs while Lavender described the ^{81m}Kr ventilation scans and ^{99m}Tc perfusion scans. The cases were in four groups: 1) patients with pulmonary emboli who showed a ventilation-perfusion mismatch, that is a normal ventilation scan, and an abnormal perfusion scan; However, if the patient has had a pulmonary infarction, both the ventilation and the perfusion scans will show defects in the same areas. 2) Patients with emphysema who showed matching ventilation and perfusion defects; 3) Patients with pulmonary consolidation owing to pneumonia (mismatched in the opposite way to those with emboli) who showed normal perfusion scans and abnormal ventilation scans; 4) Patients with lobar collapse, who showed only minimal defects on the perfusion scan in areas where there was absent ventilation. In conclusion the authors emphasized that in the diagnosis of pulmonary emboli, the chest radiograph, the ventilation, and the perfusion scans together must be analyzed for an appropriate diagnosis.

John B. West (Univ. of California, San Diego) discussed regional differences of function within the lungs. West, who is undoubtedly one of the world's leading pulmonary physiologists, first led the audience through the basic pressure relationships among the alveolar gas, the pulmonary arteries, and the pulmonary veins and then described new work in which he is currently engaged. In the upper third of the lung, commonly known as the lung apex, it is possible for pulmonary alveolar pressure to exceed pulmonary arterial pressure, which in turn exceeds pulmonary venous pressure. Under these abnormal conditions, there would be no flow in the pulmonary vascular system. One condition that can produce

such a state is shock, with pulmonary arterial hypotension and an increase in pulmonary alveolar pressure due to positive-pressure respiratory therapy. In the mid-portion of the lung under ordinary conditions, pulmonary arterial pressure exceeds pulmonary alveolar pressure, which in turn exceeds pulmonary venous pressure. It is of considerable clinical interest that in this section of the lung, in contrast to the base, pulmonary venous pressure does not determine blood flow. In the basal or bottom third of the lung pulmonary arterial pressure exceeds pulmonary venous pressure, which in turn exceeds pulmonary alveolar pressure. The reason for these pressure differences is that in the erect adult gravity causes greater pressure at the bases in both the pulmonary arteries and veins and less pressure at the apex by approximately 15 cm of water in each case.

As far as ventilation is concerned, West indicated that ventilation is also greater in the base than in the apex, but the difference between the two is not as marked as the difference in perfusion. West also reminded the audience that intrapleural pressure is more negative at the top of the thorax than at the bottom. This phenomenon occurs because the base of the lung has a smaller expanding pressure than the apex and the base a greater change in volume per unit resting volume than in the apex. The ratio of ventilation to blood flow is low at the lung base and high at the apex, which translates to a higher partial pressure of oxygen and a lower partial pressure of CO_2 at the apex. Since the lung base has a smaller expanding pressure, it was assumed that the size of each alveolus would be considerably greater at the apex. Pathologists have not found this condition, but of course the assumption is true only in the live animal. West and his group proved the point with whole lung sections made from upright frozen dogs. The alveoli were four times as large at the apex as at the base. Because the lung is suspended in the erect human, the stresses are much greater at the apex than at the base, which he related to the development of centrilobular emphysema, that is much more common at the apex.

West's current interests lie in two directions. He is trying to assess gas exchange in the lung at the height of the summit of Mt. Everest. His initial conclusion is that at times barometric pressure at this altitude is considerably greater than initially assumed, and in other respects the atmosphere on the mountain may differ from air elsewhere at the same altitude. However, this will depend to a large degree on the weather and wind conditions at the summit on any specific day. Therefore, the possibility of ascending to the summit of Mt. Everest without oxygen, accomplished recently by two Germans, would

vary from day to day.

The other line of current research West has undertaken is related to pulmonary blood flow and ventilation during weightlessness. When the space shuttle is launched, experiments on this topic will be carried out. As a pilot study, the distribution of ventilation was tested in a Lear Jet, which can achieve weightlessness for its passengers for about 25 sec by an appropriate maneuver. By using a single-breath argon-oxygen mixture, ventilation was evaluated at weightlessness. As expected, it became uniform throughout the lung instead of being greater at the base than the apex as it is under the influence of gravity. As is always the case with John West, he has the unique ability to present complex material in a lucid fashion, and the talk given to the Fleischner Society Symposium was no exception.

Symposium proceedings are not being published.